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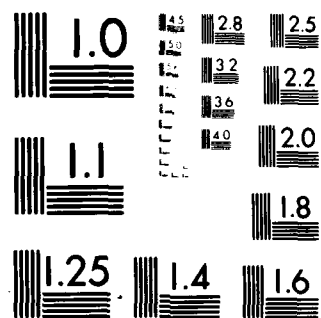
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Patients with a positive history for and laboratory testing for Hepatitis B are being denied routine dental care. This disease is caused by a detectable virus; however, there are forms of hepatitis that are caused by pathogens for which testing is not presently available. Since these patients may be clinically asymptomatic and may intentionally deny any positive past history, it becomes necessary to observe maximum precautions in sterile technique with all patients. Denying care to those individuals suspect of harboring Hepatitis B will not solve the problem.

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Introduction

Recently the Washington Post¹⁶ published a short article on the existence of the hepatitis B virus in 13% of all Southeast Asian refugees, stressing the fact that dentists should take warning of the possible danger lurking in this particular group of people who will be seeking dental care. Hepatitis B is of concern to the dentists when epidemiological statistics are examined. Dentists show a 13.6% incidence of previous exposure to the virus compared to a 2 to 5% rate in the general population.⁸ The exposure rate for oral surgeons is 21% -- four to ten times that of the normal population.¹¹ The term "carrier" also has significance with the dental population. Dentists demonstrate a 1% "carrier" rate¹⁶ and oral surgeons show a 2.3% incidence⁸ while that of the normal population is .3%.¹⁶ These statistics mean that a significant percentage of dentists harbor the hepatitis B virus particles asymptotically and have the potential of infecting patients. There are documented cases where a hepatitis B infection has been traced back to a dentist who unknowingly was a "carrier" of the virus.¹⁹ Furthermore, practices have been forced to close when hepatitis outbreaks have been traced to an infected dentist.⁸ Annually there are approximately 60,000 documented cases of hepatitis B, and this may represent only the tip of the iceberg.¹⁶

This paper will briefly discuss the pathogenesis of hepatitis B in order to appreciate the sociological and psychological implications of the disease which indicate that some patients are being delayed or denied dental care. Finally, a working solution will

be presented with suggestions for disseminating critical information concerning the management of patients with hepatitis B.

The Biology of Hepatitis

There are several viruses which are known to infect the liver; however, the hepatitis A and B viruses are the most notorious offenders.⁹

When acute hepatitis caused by these two agents is comparatively evaluated, several facts stand out. (See Appendix.) Since hepatitis B virus has a longer incubation period and a greater chance of exhibiting an anicteric state compared to the A type, these patients are more likely to be asymptomatic and, therefore, may be engaged in normal activities including routine dental treatment. The "carrier" state which has been previously mentioned is existent in only the B variety of hepatitis. In order to understand the significance of the carrier, it is necessary to have an appreciation of the morphology of the virus and the pathogenic mechanisms associated with this organism.⁹

The hepatitis B virus produces three different morphological antigens: the tubular, spherical, and Dane particles. All three forms demonstrate hepatitis B surface antigen specificity which is abbreviated to HBsAg. The Dane particle represents the virus itself, and a patient demonstrating HBsAg in the blood is considered to be potentially infective.

Once exposed to the virus, the liver becomes infected with the organism.⁹ Subsequently this agent causes the production of antigens on the surface of the hepatocytes. The response of

the host determines whether the disease will be manifested as an acute episode, a chronic infection, or a "carrier state."

In the acute form, the host responds immunologically, and the result is elimination of the virus along with damage to the infected hepatocytes. The liver, however, will soon regenerate this lost tissue and the patient will recover. The only evidence that will remain of the disease will be a persistent antibody level to the hepatitis B virus.⁹

In the patient with chronic hepatitis, the host defenses are not able to eliminate the virus. The immunological reaction may be sufficient to cause ongoing liver damage that is both progressive and greater than the rate of regeneration. Alternatively, there may be a continuing inflammatory reaction in the liver not associated with irreversible destruction of tissue, cirrhosis or other serious sequella. The regenerative capacity in this case is equal to or greater than the rate of destruction offering a better prognosis. Therefore, there are two chronic forms of hepatitis, the former being the chronic active variety and the latter being the chronic persistent type. Both forms demonstrate a persistent HBsAg, an absence of antibody to HBsAg, and an elevated SGOT, which is a serum enzyme indicating cellular necrosis in the liver. Liver biopsy is the only way to differentiate the two varieties.⁹

The "carrier state" results from failure in the immune system to react with the hepatitis B virus. There is a persistence of the organism and no evidence of liver damage. The "carrier" demonstrates the presence of the HBsAg and an absence of the antibody to this antigen; however, the SGOT in this patient is

not elevated. These people, as well as some patients with chronic hepatitis may harbor the virus asymptotically.

By comparing some of the laboratory values in each of the three states, it is possible to differentiate between a patient who may have had an acute hepatitis, is in a chronic stage, or is demonstrating a "carrier" state. The antibody to the HBsAg persists in those patients who have eliminated the virus and are recovered. The persistence of the virus manifested by an elevated HBsAg means that the Dane particle may be present and that the patient is potentially infective.⁹ The American Liver Foundation states that as little as .0004 ml of blood is infective.⁴ The virus has also been demonstrated in saliva.

With hepatitis B presenting a formidable danger, dentists are obligated to take preventive measures. The medical history may be helpful in identifying infective patients. Patients who have the following history may be suspected of harboring the virus:⁴

- | | |
|-----------------------|-------------------------------|
| 1. leukemia | 6. Down's syndrome |
| 2. blood transfusions | 7. drug addicts |
| 3. immunosuppression | 8. homosexuals |
| 4. organ transplants | 9. prisoners |
| 5. renal dialysis | 10. insitutionalized patients |
| | 11. tattoos/pierced ears. |

Goebel,⁵ however, in the June 1979 issue of the Journal of the American Dental Association brings out some interesting observations from a clinical study. He saw from the sample of patients he evaluated:

1. 56% of the patients who gave a history of hepatitis B had evidence of type B infection.
2. 58% of those with a history of type A actually had had type B
3. 51% of the patients who were carriers gave negative histories.

From the above data, Goebel⁵ concluded that patients did not know consistently with what type of hepatitis they had been infected, and that the medical history was unreliable for screening for this disease.

Based upon the literature cited in the previous discussion, the values for HBsAg, HBsAg antibody, and SGOT may offer a method for laboratory screening. Hribar,⁷ however, states that the best available tests for hepatitis B could only detect 20 to 50% of the "carriers."

" Sociological/Psychological Effects of Hepatitis B

The asymptomatic carrier of the surface antigen of hepatitis B presents a danger to both the dentist and the patient. In 1975, a Los Angeles dentist who unknowingly harbored the virus was found to have infected 15 patients. He subsequently was barred from practice by county health authorities.¹⁴ Dr. James Mosley,¹⁴ Director of Hepatic Epidemiology at Los Angeles County's John Wesley Hospital, stated at that time that, "health professionals with active disease should stop practice."

With the threat of losing the right to practice, the results of a study recently conducted by Powers et al.¹⁸ are not surprising. Thirty dentists rated 56 potentially stress provoking situations.

The threat of contracting hepatitis ranked third. It was ahead of dealing with a medical emergency in the operatory, and of the dentist himself having chest pains. Since a dentist with active hepatitis can be legally stopped from practicing, the authors interpreted the findings on hepatitis as being consistent with the threat to financial security as well as personal health.

The hepatitis B issue has recently received some publicity in the Washington Post in connection with the influx of Southeast Asian refugees. In her article, Susan Okie¹⁶ states that Maryland dentists are now being warned by the State Health Department that they risk getting hepatitis if they do dental work on Indochinese refugees who have not been tested for the disease. Since these people have a 12% "carrier" rate compared to a .3% rate for the general population in the United States, a greater risk exists to practitioners who must come in contact with the blood and saliva of their patients.¹⁶ Dr. Phillip A. Pushkin,¹⁶ Director of Dentistry for Baltimore County in Maryland states, "When the refugees hit this country, the first thing they do is ask for a dental appointment; dentists should refuse to treat refugees until they have had a blood test for hepatitis. If a dentist becomes a 'carrier,' his career may well be over." It is not surprising that some dentists are requiring serological screening for hepatitis B for all patients prior to treatment⁶ and that some practitioners are refusing to treat the infectious patients referring them to hospitals or teaching centers with facilities for managing this problem.¹⁴

Institutional dentistry is not anxious to handle the hepatitis issue and this observation is reflected in the experience of Martha Newell,¹⁵ a volunteer worker for the Center for Southeast Asian Refugees in Washington, D.C. She says that these people are in great need of dental care when they arrive in this country and that it has been difficult to obtain routine care from local dental schools.

During a recent interview with a spokesman from a state dental association it was disclosed that this organization was unaware of anyone having difficulty obtaining treatment who had a persistent surface antigen for hepatitis B. However, according to the chief of the diagnostic clinic in this state's dental school, no patient with "active hepatitis" would be given routine dental care at that institution. "Active hepatitis" was defined as a persistent surface antigen for hepatitis B.

On the basis of the above interviews, a telephone survey was conducted with 23 of the 59 U.S. dental schools to find out what percentage were rendering routine dental care to those patients with a persistent surface antigen for hepatitis B. Although all schools provided emergency treatment, 11 schools (47% of the sample) did not provide routine dental services to these patients. One institution said that one of their dental hygiene students had contacted the disease recently and now was at 8% liver function.²¹

From the above information, it is clear that a persistent hepatitis B surface antigen presents a problem for both the patient and practitioner. The threat of being barred from practice is

a financial concern for the dentist. Patients with the problem are being denied routine dental care and are only able to be seen on an emergency basis. Additionally, it can be speculated that those who have not been given routine dental care because of the persistent antigen may be tempted to not report any history of hepatitis to the next dentist.

Solution

Part of the answer to the hepatitis issue rests with dental schools since they are responsible for educating students and often provide the bulk of updated information to graduate practitioners through continuing education. Even though the A.D.A. states that this problem can be managed in a normal clinical setting, there is proof that many dental schools are not providing routine care to these patients.²¹ By avoiding the hepatitis issue, these institutions are not providing their students with the education necessary to manage hepatitis patients. For now, the names of those facilities who do provide care should be publicized so that these patients can be properly managed.

Current recommendations for the hepatitis B patient appeared in the 11 January 1980 issue of the Morbidity Mortality Weekly,¹² and were the result of a joint effort by the American Dental Association and the Center For Disease Control. It is unlikely that most dentists have access to this publication and certainly such information should be in more widely circulated literature. Periodicals, dental schools, and continuing education curricula should disseminate the most current information on hepatitis B.

Once dentists are aware of the hazards to their own health, and of the proper management of hepatitis patients, they will be able to manage these patients confidently and effectively, and no one will be denied treatment.

The medical history and necessary laboratory tests aid the clinician in identifying many problems in the patient. With hepatitis B, these diagnostic tools are of dubious value. The medical history is far from being a reliable screening method;⁵ laboratory tests are only 20-50% accurate,⁷ and currently there are other forms of hepatitis for which there are no tests.² Rather than sequestering these patients with a persistent antigen from the avenue of routine care, elevating the standards for sterile technique in all phases of clinical dentistry for every patient may be the answer to protection for the practitioner.

Autoclaving and proper disinfection are obvious prerequisites to practicing aseptic dentistry. The break in sterile technique, however, probably stems from another source. In a study conducted by Bancroft *et al.*,² it was seen that a parenteral portal of entry was necessary to establish a hepatitis B infection. It would seem that dentists need not worry if there are no cuts or nicks on their hands or fingers. However, in a study by Allen¹ it was seen that traces of occult blood can be found under the fingernails of barehanded dentists after performing seemingly bloodless dental procedures. Not only might there be microscopic abrasions, but the nail beds may offer a means for passing the virus on to other patients. Feldman and Schiff,³ in 1973, reported that the majority of dentists do not wear gloves. In 1977, 55 cases of

hepatitis B were traced to a single oral surgeon who was found to be an asymptomatic carrier. This practitioner never wore gloves and admitted repeatedly cutting his fingers while working in patients' mouths.¹⁹ From the above investigations it can be seen that the use of gloves may reduce the risk of exposure to hepatitis B and to other diseases which are equally formidable.

The burden in this issue rests in disseminating the necessary information to the practitioners. Until this is effectively done, the dentist either will be oblivious to the hazards of hepatitis B or consider refusing to treat those individuals suspected of harboring the antigen. Once denied treatment, the patient is tempted to falsify the medical history with the next dentist rather than actively seek care from a practitioner who can intelligently manage this situation. This increases the risk to those dentists who do not take adequate precautions and to their non-infected patients.

Conclusion

From the telephone survey with dental schools, it becomes clear that the health history and laboratory screening are used to determine what level of dental care some patients will receive. As far as hepatitis is concerned, these methods will not detect everyone who harbors the virus and certainly they are no substitute for sound aseptic standards. The answer to this issue lies in educating the profession to upgrading their sterile technique to meet the threat of this disease and other equally dangerous pathogens which may not be detectable with present methods.

Appendix

FEATURES	TYPE A	TYPE B	TYPE NON-A, NON-B
Incubation period	About 25 days; mean range, 15-40 days	About 70 days; mean range, 50-180 days	About 60 days, mean range, 15-180 days
Type of onset	Usually acute	Usually insidious	Usually insidious
Fever	Common; precedes jaundice	Less common	Less common
Age group affected	Usually children and young adults	All age groups	All age groups
Prodrome: arthritis and rash	Not present	May be present	Unknown
Jaundice	Rare in children; more common in adults	Rare in children; more common in adults	Same as types A and B
Abnormal SGOT or SCPT	Transient-1 to 3 weeks	More prolonged-1 to 8+ months	Prolonged
Thymol turbidity	Usually increased	Usually normal	?
IgM levels	Usually increased	Usually normal	?
HBsAg (Australia antigen) in blood	Not present	Present in incubation period and acute phase; occasionally may persist	Not present
Virus in feces	Present during late incubation period and acute phase	May be present but no direct proof	Unknown
Virus in blood	Present during late incubation period and early acute phase	Present during late incubation period and acute phase; occasionally persists for months and years	Probably like type B
Carrier state	No	Yes	Probably yes
Immunity			
Homologous	Present	Present	Unknown
Heterologous	None	None	Unknown

Taken from Kugman and Gocke⁹ with permission from the publisher.

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